Please substitute the following set of claims for the pending claim set.

IN THE CLAIMS

1-59. (Canceled)

60. (Currently Amended) A hypermutable, transgenic mouse wherein the germ and somatic cells of said mouse express a transgenic polynucleotide encoding a dominant negative form of a human PMS2 mismatch repair protein, wherein the protein comprises the first 133 amino acids of human PMS2.

61. (Currently Amended) A hypermutable, transgenic mouse produced by a process comprising the steps of:

introducing a transgenic polynucleotide encoding a dominant negative form of a ~~*PMS2*~~ human PMS2 mismatch repair protein into a fertilized mouse egg, wherein the protein comprises the first 133 amino acids of human PMS2, whereby said protein is expressed and said fertilized mouse egg becomes hypermutable;

implanting the fertilized egg into a pseudopregnant female; and

allowing said mouse egg to develop into a hypermutable, transgenic mouse.

62. (Currently Amended) A method of making a hypermutable fertilized mouse egg comprising:

introducing into said fertilized mouse egg a transgenic polynucleotide encoding a dominant negative form of a ~~*PMS2*~~ human PMS2 mismatch repair protein, wherein the protein comprises the first 133 amino acids of human PMS2, whereby said protein is expressed and said fertilized mouse egg becomes hypermutable.

63-70. (Canceled)

71. (Currently Amended) A method for generating a mutation in a gene of interest comprising the steps of:

introducing a transgenic polynucleotide encoding a dominant negative form of a ~~*PMS2*~~ human PMS2 mismatch repair protein into a fertilized mouse egg, wherein the protein comprises the first 133 amino acids of human PMS2, whereby said protein is expressed and the fertilized mouse egg becomes hypermutable;

implanting the fertilized egg into a pseudopregnant female;

allowing said fertilized mouse egg to develop into a hypermutable, transgenic mouse; and

testing the mouse to determine whether the gene of interest harbors a mutation.

72. (Previously Presented) The method of claim 71 wherein the step of testing comprises analyzing a nucleotide sequence of the gene of interest.

73. (Previously Presented) The method of claim 71 wherein the step of testing comprises analyzing mRNA transcribed from the gene of interest.

74. (Previously Presented) The method of claim 71 wherein the step of testing comprises analyzing a protein encoded by the gene of interest.

75. (Previously Presented) The method of claim 71 wherein the step of testing comprises analyzing the phenotype of the gene of interest.

76-81. (Canceled)

82. (Currently Amended) The method of claim [[81]] 62 wherein said dominant negative form of a human PMS2 mismatch repair protein is encoded by a polynucleotide which comprises a truncation mutation at codon 134 of SEQ ID NO:1.

83. (Previously Presented) The method of claim 82 wherein the truncation mutation is a thymidine at nucleotide 424 of wild-type *PMS2* of SEQ ID NO:1.

84-85. (Canceled)

86. (Previously Presented) The hypermutable, transgenic mouse of claim 61 wherein the transgenic polynucleotide comprises a truncation mutation at codon 134 of SEQ ID NO:1.

87. (Previously Presented) The hypermutable, transgenic mouse of claim 86 wherein the truncation mutation is a thymidine at nucleotide 424 of wild-type *PMS2* of SEQ ID NO:1.

88. (Canceled)

89. (Currently Amended) The mouse of claim [[88]] 60 wherein said transgenic polynucleotide comprises a truncation mutation at codon 134 of SEQ ID NO:1.

90. (Previously Presented) The mouse of claim 89 wherein the truncation mutation is a thymidine at nucleotide 424 of wild-type *PMS2* of SEQ ID NO:1.

91. (Canceled)

92. (Currently Amended) The method of claim [[91]] 71 wherein said transgenic polynucleotide comprises a truncation mutation at codon 134 of SEQ ID NO:1.

93. (Previously Presented) The method of claim 92 wherein the truncation mutation is a thymidine at nucleotide 424 of wild-type *PMS2* of SEQ ID NO:1.

94-96. (Canceled)